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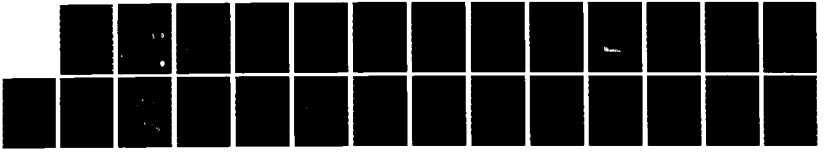

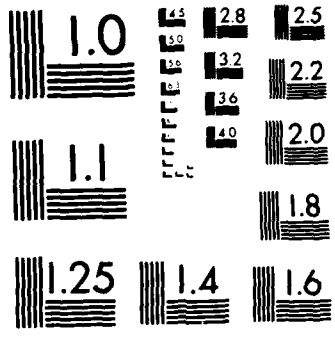

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VESTIBULAR SYSTEM PHYSIOLOGY AND SPACE MOTION SICKNESS: AN INTRODUCTION

Dean L. Kellogg, Jr., M.D.

Department of Physiology
University of Texas Health
Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, TX 78229

August 1986

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This report has been reviewed and is approved for publication.

DAVID R. JONES, M.D.
Project Scientist

JAMES R. HICKMAN, Jr., Col, USAF, MC
Supervisor

JEFFREY G. DAVIS, Colonel, USAF, MC
Commander

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The purpose of this report is to give the reader a basic understanding of the relationships between the vestibular system and space motion sickness. Space motion sickness is reviewed from a historical perspective. The symptomatology and course are described. The vestibular system anatomy and basic physiology are outlined. Two theories of space motion sickness are presented. Finally, the results of several experimental investigations completed over the last 10 years are examined to expand the reader's understanding of space motion sickness.

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VESTIBULAR SYSTEM PHYSIOLOGY AND SPACE MOTION SICKNESS: AN INTRODUCTION

INTRODUCTION

The purpose of this report is to give the reader a basic understanding of the relationships between the vestibular system and space motion sickness. Space motion sickness is reviewed from a historical perspective. The symptomatology and course are described. The vestibular system anatomy and basic physiology are outlined. Two theories of space motion sickness are presented. Finally, the results of several experimental investigations, completed over the last 10 years, are examined to expand the reader's understanding of space motion sickness.

HISTORICAL PERSPECTIVE

Man first encountered space motion sickness on 6 April 1961, during the 17 orbit flight of Gherman Titov aboard Vostok 2 (13). Upon entering his first orbit, Titov reported the following:

> "I had the sensation that I was assuming a head-down position. Objects that surrounded me seemed to float upwards, and only after one or two minutes did they seem to return to their places."

He reported no further difficulties until during his 7th orbit. At that time, he reported the following:

> "I felt a certain discomfort, similar to the first symptoms of seasickness. It was mostly felt during abrupt movement of the head, though this sensation did not affect my efficiency. I felt giddy and nauseated. This was accompanied by a deterioration of the appetite. Falling asleep was a bit difficult."

This unpleasant sensation of nausea continued until he began to encounter increased gravitational (G) forces during reentry.

The numbers of astronauts and cosmonauts who suffered from space motion sickness and the spacecraft they flew, up to and including the 3rd Space Shuttle mission, are shown in Table 1 (9).

Note that Vostok/Voskhod, Salyut-6, Apollo, Skylab, and the Shuttle all had sick crewmembers, while Soyuz, Mercury, and Gemini had none. The lack of sickness has been attributed to three factors: (1) the small size of the vehicles' crew compartments; (2) the crewmen tended to wear their helmets more in these vehicles, thus restricting their head movements; and (3) they tended to remain restrained in their seats more than in the other craft, thus restricting their body movements. Overall, since Titov's flight, 50%-60% of the astronauts and cosmonauts have suffered some manifestations of space motion sickness.

Besides the sensory illusions reported by Titov, a number of other symptoms occur in space motion sickness. These symptoms include nausea, vomiting, cold sweating, increased salivation, drowsiness, headache, dizziness, skin pallor or flushing, and subjective warmth.

The time course for space motion sickness over the first 14 hr during an early Space Shuttle mission, using a subjectively determined "overall discomfort" scale, is shown in Figure 1. Note that, within the first hour in orbit, this astronaut began to feel slight discomfort. At approximately 4 hr into the mission, he vomited. He vomited twice more before taking an antimotion sickness medication (a scopolamine/Dexedrine combination) 6.5 hr into the mission. The medication gave some relief, but at 9.5 hr he vomited again, despite being in the presumed period of scopolamine/Dexedrine effectiveness. If we were to follow this astronaut's sickness over the course of the ensuing 4 to 6 days in orbit, we would find a decreased frequency of vomiting and a gradual decrease in overall discomfort. All symptoms were resolved by the end of 4 to 6 days in orbit (15).

Though some public attention has been drawn to the space motion sickness problems, it is not generally appreciated that space motion sickness has decreased astronaut performance, especially during early mission stages. In one particularly severe case, a Soviet mission was aborted after 2 days in orbit. So, in susceptible individuals, space motion sickness develops rapidly upon entering weightlessness. The severity of symptoms declines over 4 to 6 days, after which it is completely resolved.

VESTIBULAR SYSTEM STRUCTURE AND FUNCTION

A diagram of the membranous labyrinth, including the peripheral vestibular apparatus, is shown in Figure 2. The apparatus consists of 2 types of receptor organs: the otolith organs, which consist of the saccule and utricle, and the semicircular canals. The entire membranous labyrinth is filled with endolymph fluid, which is much like intracellular fluid in its ionic composition. The membranous labyrinth itself is suspended in the bony labyrinth and is bathed in perilymph fluid, which is much like extracellular fluid in its ionic composition. The peripheral vestibular apparatuses are located in the petrous portion of the temporal bones bilaterally and are innervated by the VIIIth cranial (acoustic) nerve (11).

The otolith organs, the saccule and utricle, play important roles in transducing information about head position relative to gravity and active head tilt. The otolith organs sense linear accelerations, including gravitational acceleration. A linear acceleration can be defined as a change in the velocity of an object traveling along a straight line. Each otolith organ contains a specialized patch of mechanoreceptor hair cells, known as the maculae. A diagram of a utricular macula is shown in Figure 3 (11). Saccular maculae have a similar anatomy. A macula consists of crystalline otoconia loosely embedded in a gelatinous membrane. This membrane overlies a specialized sensory epithelium consisting of type I and type II hair cells which are surrounded by supporting cells. Both types of hair cells have apical projections. These projections are long kinocilia and shorter stereocilia filaments on the free surface of a cell. Each individual hair cell has 1 kinocilium and numerous stereocilia. The kinocilium represents both a morphological and

TABLE 1. NUMBER OF ASTRONAUTS/COSMONAUTS SUFFERING MOTION SICKNESS IN SPACE

Spacecraft	Total No. of Persons Flown	No. Sick
Vostok/Voskhod	11	4
Soyuz	13	0
Salyut-6	27	12
Mercury	6	0
Gemini	16	0
Apollo	33	11
Skylab	9	5
Space Shuttle	8	4

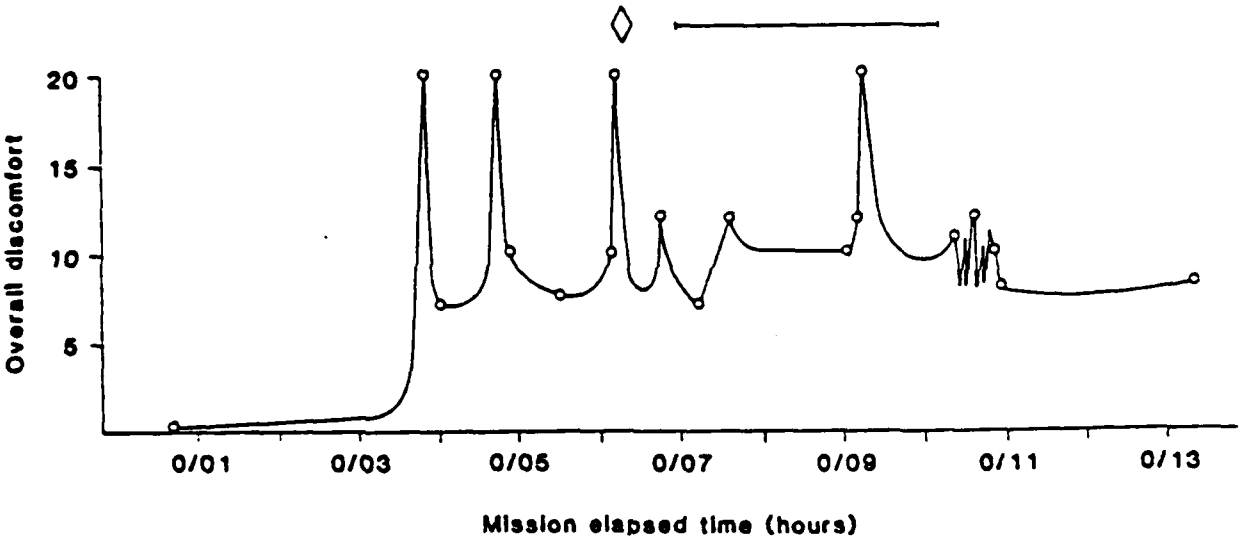

Figure 1. Magnitude estimate of discomfort (2) for 1 subject during the first 14 hr in orbit. A score of 20 indicates vomiting. Curves between data points were interpolated by the subject. Diamond represents medication (scopolamine and Dexedrine), followed by horizontal bar representing period of presumed maximal effectiveness.

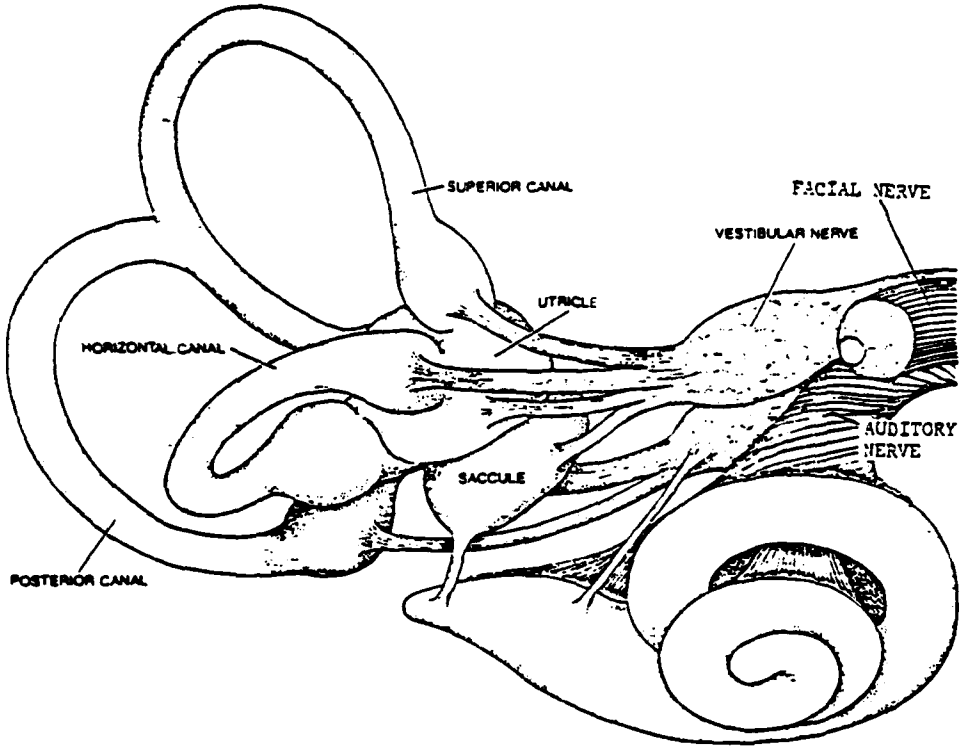

Figure 2. Diagram of the membranous labyrinth, including the peripheral vestibular apparatus.

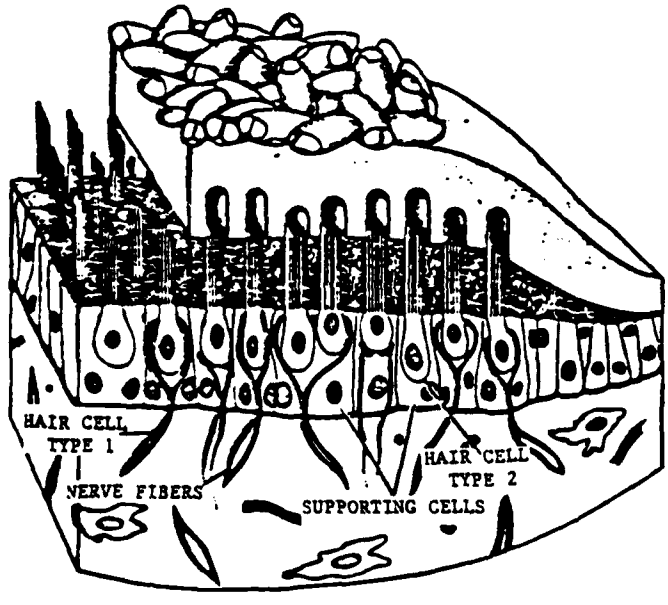

Figure 3. Diagram of a utricular macula.

functional polarization of the cell. Afferent and efferent fibers of the VIIIth cranial nerve innervate the hair cells. With the head in a vertical position, the utricular maculae are oriented horizontally and the saccular maculae are oriented vertically. The otolith organs function as follows:

1. Otoconia act as an inertial mass which resists the application of external forces.

2. When a linear acceleration is applied to the head, the otoconia and their gelatinous membrane tend to stand still, while the hair cell patch slides underneath.

3. The linear acceleration creates a shearing force which bends the kinocilia and stereocilia of the hair cells.

If a hair cell's stereocilia are bent in the direction of the cell's kinocilium, the hair cell is depolarized. This depolarization leads to increased firing of the afferents from that hair cell. If the stereocilia are bent away from the kinocilium, the hair cell is hyperpolarized. This hyperpolarization leads to decreased firing nerve fibers. The overall hair cell population of an otolith macula is arranged to take advantage of this hair cell polarization, as shown in Figure 4. The figure also shows the distribution of hair cell polarization axes in a utricular macula. Again, saccular maculae would be similar. The head of each arrow can be thought of as representing a hair cell kinocilium, while each arrow's tail represents stereocilia. Note that the hair cell's polarization axes are directed toward a macular region known as the striola. Also notice that the hair cell axes do not make a uniform pattern (5, 11).

The arrangement allows the otolith organs to respond to head tilt or linear accelerations in any one of several directions, by producing a unique pattern of hair cell depolarizations and hyperpolarizations across the macula. In the 1 G environment of Earth, the otolith organs give tonic inputs to higher neural center because they are constantly stimulated by gravity. The organs respond tonically to head position relative to Earth's gravity. Otolith organs will respond phasically to other transient linear accelerations.

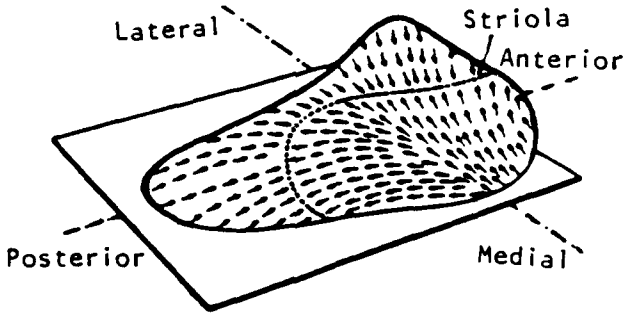

Figure 4. Hair cell polarization.

In the microgravity conditions of orbit, the otolith organs respond phasically to such transient accelerations, but being out of their normal 1 G environment, they no longer give any tonic inputs. The organs can no longer signal head position.

The semicircular canals are quite different; they respond to angular accelerations. Angular accelerations can be defined as changes in circular motion velocity, as when the head is turned left or right. The semicircular canals transduce information about changes in head rotation rate and direction. The 3 canals are arranged to give information on angular accelerations in the X, Y, and Z axes in three-dimensional (3-D) space (5).

In each semicircular canal, mechanoreceptive hair cells are found in a specialized structure known as the ampulla. Oblique and transverse views of an ampulla are shown in Figure 5 (11).

These hair cells are anchored in a region known as the crista. Their kinocilia and stereocilia are embedded in a diaphragm-like gelatinous membrane known as the cupula. The cupula bisects the ampulla. Where in the otolith organs the inertial mass was provided by the otoconia, in the canals it is provided by the endolymph fluid. The semicircular canals function as follows:

1. When the head is rotated, the endolymph tends to remain "stationary" due to inertia.

2. The cupula, along with kinocilia and stereocilia embedded in it, is displaced by the "stationary" endolymph in a direction opposite to the head rotation. This displacement causes a shear force on the kinocilia and stereocilia, forcing them to bend.

3. The bending hyperpolarizes or depolarizes the hair cells, and VIIIth cranial nerve afferent activity is altered accordingly, as we saw for the otolith macula.

In a 1 G environment, the semicircular canals respond to transient angular accelerations and are not affected by gravity. So, in the microgravity of orbit, the canals should function just as they do on Earth in 1 G (5, 11).

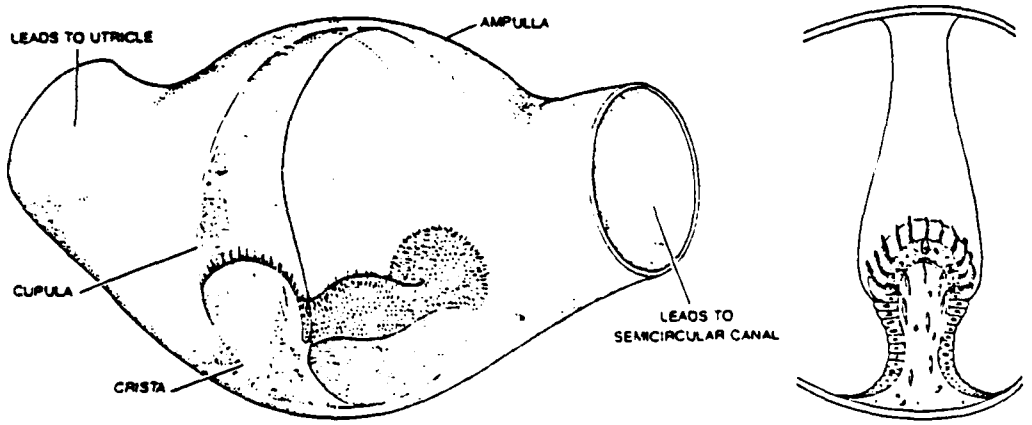

Figure 5. Oblique and transverse views of an ampulla.

The neural pathways which connect the peripheral vestibular apparatus with the central nervous system (CNS) are quite complex, as shown in Figure 6 (2).

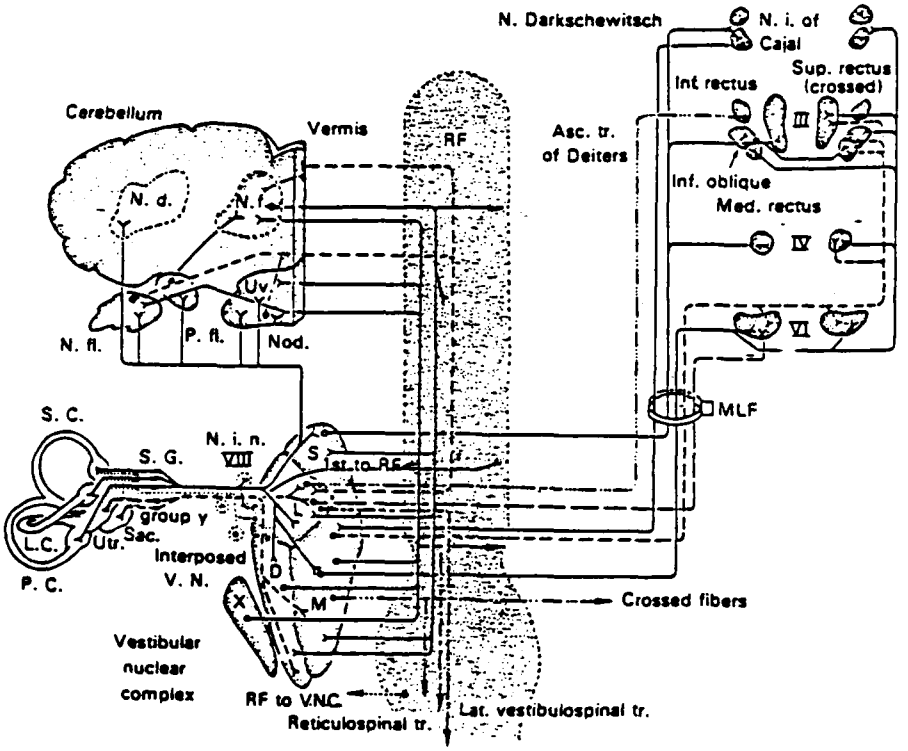

Figure 6. Neural pathways connecting the peripheral vestibular apparatus with the central nervous system.

The specific pathways shown are obviously complex. However, for our purposes, these pathways can be simplified and presented as 5 basic projections:

1. Periphery to vestibular nuclear complex,

2. Projections to cerebellum,

3. Projections to oculomotor nucleii via the Medial Longitudinal Fasciculous (MLF),

4. Projections to spinal cord via vestibulospinal tracts, and

5. Projections to Reticular Formation (RF).

None of these known pathways, individually or together, can account for the symptomatology of space motion sickness or even general motion sickness (8).

From work on humans and animals, it has been determined that 3 structures are necessary for motion sickness development:

1. A functioning labyrinth,

2. The chemotactic trigger zone (or area postrema), and

3. The vomiting integration center.

Presently, how these structures interrelate in motion sickness is unclear.

Contrasting this uncertainty is our relatively good understanding of the vestibular system's role in orientation and balance. A diagram of a proposed "Orientation and Balance System" is shown in Figure 7 (11).

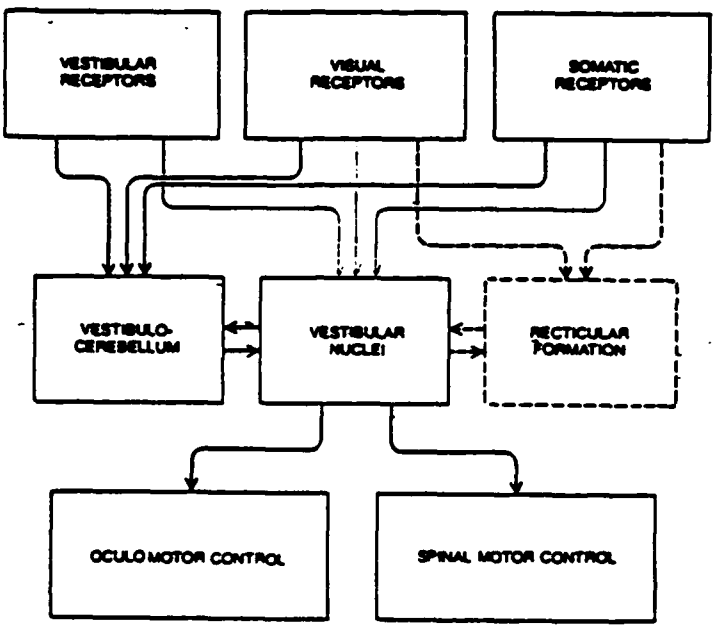

Figure 7. Orientation and balance system.

The system functions as follows:

1. Sensory inputs from vestibular, visual, and perhaps somatic receptors are fed into central areas.

2. The central areas (the vestibulocerebellum, vestibular nuclei, and perhaps reticular formation) integrate these sensory inputs.

3. The output from the central integration areas serves to coordinate body and head movements and orientation in 3-D space.

The concept of sensory integration systems such as this one has been used as a basis for 2 general theories of motion sickness: (1) the Overstimulation/ Fluid Shift Theory, and (2) the Sensory Conflict Theory. These theories have also been applied to space motion sickness (11).

THEORIES OF MOTION SICKNESS

The first theory is the Overstimulation/Fluid Shift Theory (7). This theory proposes that motion sickness results from an overstimulation or irritation of the vestibular system. This overstimulation causes a "spillover" or "leakage" of neural activity from balance and orientation centers to other

in turn give rise to the symptoms of motion sickness. The Overstimulation Theory has been applied to space motion sickness as follows:

1. Upon entering 0 G, there is a fluid shift from dependent body to the upper body.

2. This fluid shift may then either raise intracranial pressure, leading to space motion sickness symptoms, or may cause an acute Meniere's disease-like picture.

3. In the latter case, the fluid shifts would increase perilymphatic and/or endolympathic pressures. This increased pressure would irritate the peripheral vestibular apparatus, causing overstimulation of CNS centers, neural spillover, and, finally, space motion sickness symptoms.

4. According to this view, recovery from space motion sickness in orbit is due to reestablishment of proper fluid and pressure balance.

The second theory of motion sickness is the Sensory Conflict Theory (7). According to this theory, an asynchrony or novelty of sensory inputs to CNS sensory integration centers develops first. These asynchronous inputs lead to an attempt at reorganization of spatial perceptions at the integration sites. This reorganization attempt, either by its own neural activity or by passing larger-than-normal amounts of perceptual information to other CNS locations, creates a state of increased vigilance. This vigilance in turn leads to the nausea and vomiting of motion sickness, just as heightened vigilance can lead to nausea and vomiting in students before examinations or seminars. According to the Sensory Conflict Theory, recovery from motion sickness results from adaptation of central integration centers to novel or asynchronous sensory inputs. In the case of space motion sickness, the Sensory Conflict Theory applies as follows:

1. Zero G creates novel sensory inputs from the peripheral vestibular apparatus.

2. The novel inputs lead to reorganization attempts in central integration centers.

3. The reorganization attempt creates increased vigilance, leading to space motion sickness symptomatology.

4. Recovery from space motion sickness occurs when the reorganization is complete.

EXPERIMENTAL WORK OF THE 1970s

The first detailed American experiments on human vestibular function in orbit were carried out aboard Skylab in the early 1970s. Experiment M-131 was coordinated by Ashton Graybiel of the Naval Aerospace Medical Laboratory at Pensacola, Florida (1). The M-131 experiment was designed to look at how weightlessness changed human susceptibility to vestibular-induced motion sickness.

The experiment was performed using a rotating chair (Fig. 8) (11). A stressful motion environment was created by having a seated, restrained astronaut execute head movements while being rotated in the chair. The chair could be rotated at speeds up to 30 rpm in a clockwise or counterclockwise direction.

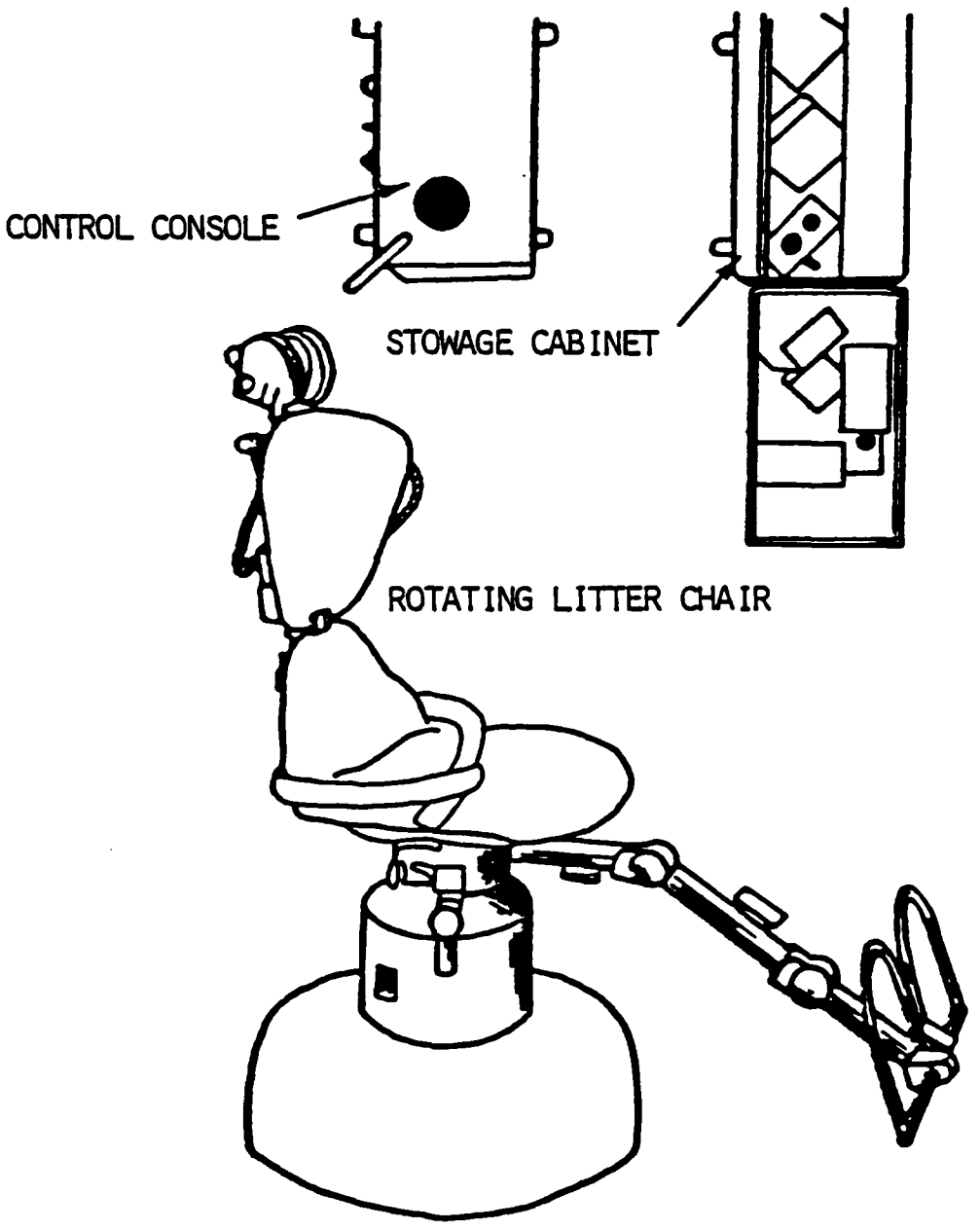

Figure 8. The rotating litter chair motion sickness test mode.

The eyes were covered at all times. The head movements were through an arc of 90° in 4 directions: forward, backward, left and right, and return to upright position. Each position was held for 1 sec except for return, which was held for 20 sec between each head movement set. The movements were executed either with the chair stationary as a control, or with it rotating. Two possible endpoints were chosen as follows:

1. 150 consecutive head movement sets without developing motion sickness, or

2. Moderate motion sickness development (operational requirements prevented going further).

Table 2 shows how motion sickness was quantified for the study (12).

TABLE 2. DIAGNOSTIC CATEGORIZATION OF DIFFERENT LEVELS OF SEVERITY OF ACUTE MOTION SICKNESS

	Levels of Severity Identified by Category				
Category	Pathognomonic 16 pts	Major 8 pts	Minor 4 pts	Minimal 2 pts	AQS[a] 1 pt
Nausea syndrome	vomiting or retching	nausea II, III[b]	nausea I	epigastric discomfort	epigastic awareness
Skin		pallor III	pallor II	pallor I	flushing/ subjective warmth >II
Cold sweating		III	II	I	
Increased salivation		III	II	I	
Drowsiness		III	II	I	
Pain					headache
Central nervous system					dizziness eyes: closed>II open III

Levels of Severity Identified by Total Points Scored				
Frank Sickness (S)	Severe Malaise (M III)	Moderate Malaise (M IIA)	Moderate Malaise (M IIB)	Slight Malaise (M I)
16 pts	8-15 pts	5-7 pts	3-4 pts	1-2 pts

[a] AQS = Additional Qualifying Symptoms
[b] Levels of severity: III = severe or marked, II = moderate, I = slight
Pts = points

The logic behind the experimental design was as follows:

1. Restraints would control for body proprioceptive inputs.

2. Blindfolding would control for visual inputs.

3. Head movement sets would cause abnormal vestibular stimulation during rotation. Any sickness produced should be due to the abnormal inputs from the otoliths and semicircular canals.

4. Head movement sets without rotation would create normal semicircular canal inputs, so any symptoms then produced had to come from abnormal otolithic function.

The results for the Skylab 3 crew are shown in Figure 9. Note the following:

1. Preflight, all crewmen were susceptible to motion sickness before 150 head movement sets were completed.

2. During flight, all crewmen reached 150 sets easily. This indicates decreased motion sickness susceptibility in orbit.

3. Postflight, all crewmen had decreased susceptibility during the first few days. However, by day 5, they were becoming susceptible, and by day 17, they were at preflight susceptibility levels.

Two weaknesses exist in the data collected: (1) the flight data were not recorded until day 5, and then were collected without rotation (remember, space motion sickness lasts 4 to 6 days); and (2) the postflight data on R+1 were collected without rotation. Data from Skylab 2 and 4 were similar and had the same weakness.

The final interpretation of the M-131 results was as follows:

1. All crewmen were susceptible to motion sickness from abnormal vestibular stimulation preflight.

2. In flight, an adaptation occurred such that the abnormal vestibular stimulation no longer induced motion sickness.

3. Postflight, this adaptation persisted for a short time, after which a readaptation to the preflight state occurred.

Because of the persistence of the adaptation after flight when there were no fluid balance shifts, the results seem to fit the Sensory Conflict Theory predictions better than those of the Overstimulation/Fluid Shift Theory. The adaptation was viewed as probably having occurred in the CNS integration centers, such that vestibular inputs to the integration centers were "downgraded" in importance relative to other sensory inputs. This adaptation happened early in flight, prior to the initial inflight trial. The adaptation persisted temporarily in 1 G, but was gradually lost as the sensory integration centers readapted to the normal 1 G condition (1, 4).

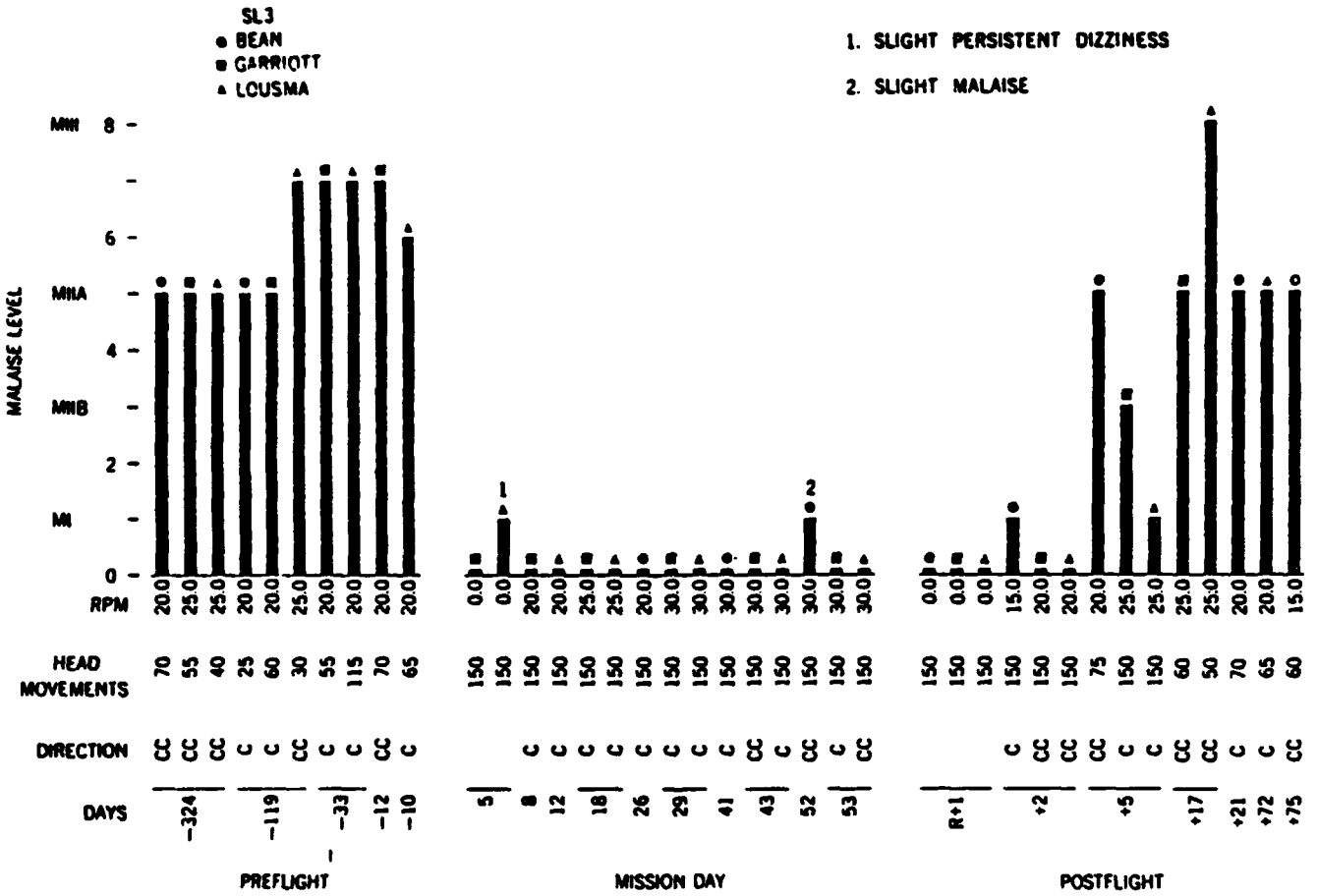

Figure 9. Motion sickness symptomatology of Skylab 3 astronauts quantitatively expressed in terms of malaise level as evoked by the test parameters (rotational velocity, number of head movements, and direction of rotation) used before, during, and after the Skylab 3 mission.

Further work in the late 1970s also supported the Sensory Conflict Theory and weakened the Overstimulation/Fluid Shift Theory in regard to space motion sickness. Parker took continuous measurements of intralabyrinthine perilymph pressures and cerebrospinal fluid (CSF) pressures in anesthetized guinea pigs and monkeys. These measurements were made while the animals' torsos and hindquarters were lifted to induce cephalad fluid shifts. The Overstimulation/Fluid Shift Theory would predict progressive elevation and imbalance of intralabyrinthine and/or CSF pressures with cephalad fluid shifts. Parker found no such pressure abnormalities with up to 12 hr of continuous hindquarters elevation. While this work can be criticized in that perilymphatic pressures and not endolymphatic pressures were taken, his work did not support the Overstimulation/Fluid Shift Theory (10).

In the late 1970s, Graybiel performed Earth-bound experiments very similar to the M-131 Skylab experiment. He looked at differences in motion sickness susceptibility in 14 subjects for 2 conditions during rotation at 30 rpm in a 10° head-down position. In condition 1, the subject was placed in a 10° head-down tilt for 15 min prior to head-down rotation, while in condition 2, the subject was 10° head-down for 6 hr before head-down rotation. Fluid shifts were verified by vital capacity measurements. Test results are shown in Table 3. Motion sickness was quantified using the point system seen in Table 2.

Graybiel found no statistical difference between condition 1 and condition 2 in terms of grouped subject data. However, when intraindividual conditions 1 and 2 results were analyzed with a paired, 1-tailed T-test, 15 min of head-down tilt was statistically worse than 6 hr of prerotation head-down tilt. Thus, Graybiel found exactly the opposite of what would be expected from the Overstimulation/Fluid Shift Theory. At the present time, as a result of Parker's and Graybiel's work, the Overstimulation/Fluid Shift Theory has been severely weakened (3).

RECENT EXPERIMENTAL WORK

With the advent of the Space Shuttle and Spacelab, new inflight work has been done on the space motion sickness problem. Much of the work of Spacelab 1 was devoted to this problem. Spacelab 4 also worked on the space motion sickness question.

The results from the Spacelab 1 experiments are still being published. Two of the many ongoing investigations are examined next, to show where current research is heading.

The first Spacelab experiment investigated visual-vestibular interactions. When an Earth-bound subject views a wide-field display screen rotating around his roll axis, he normally perceives a sensation of continuous self-rotation in a direction opposite to the field motion. This display is called "circularvection." The subject also perceives a paradox that his body is tilting at an angle opposite to the field rotation. These effects have been interpreted as being due to visual-otolithic sensory mismatch.

In the Spacelab 1 study, the astronauts viewed a polka-dotted pattern on the inside of a dome. This pattern served as a wide-field display scene. The

TABLE 3. MOTION SICKNESS POINTS AS A FUNCTION OF ELAPSED TIME FOR IMMEDIATE ROTATION (15 MIN) AND RECUMBENCY BEFORE ROTATION (6 HR) CONDITIONS

A. Condition 1 (15 min)

Subject	Elapsed time of rotation (min) 1-10	11-20	21-30	31-40	41-50	51-60	Total points
AA_1	5	7	N(20 min)				7
SB_1	0	5	5	5	8	N(53 min)	8
RB_1	0	FS(16 min)					16
FC_1	0	0	0	0	0	0	0
WF_1	4	N(14 min)					9
JH_2	2	4	FS(23 min)				16
WH_1	0	0	0	0	0	0	0
GH_2	0	0	0	3	3	3	3
BM_2	1	3	6	N(38 min)			6
JR_2	3	0	2	5	FS(43 min)		16
RS_1	0	0	0	0	0	0	0
JT_2	0	4	N(27 min)				4
SW_2	0	0	0	0	0	0	0
LW_2	2	2	4	4	4	4	4

B. Condition 2 (6 Hr)

AA_2	0	0	3	3	3	3	3
SB_2	2	0	0	0	0	2	2
RB_2	0	8	8	N(31 min)			8
FC_2	0	0	0	0	0	0	0
WF_2	1	2	3	5	5	N(52 min)	5
JH_1	0	0	0	0	0	0	0
WH_2	0	0	0	0	0	0	0
GH_1	4	9	N(26 min)				9
BM_1	0	0	3	5	N(41 min)		5
JR_1	1	3	3	8	N(40 min)		8
RS_2	0	0	0	0	0	0	0
JT_1	0	4	7	N(37 min)			7
SW_1	0	0	0	0	0	0	0
LW_1	0	0	0	0	0	0	0

Abbreviations: N = nausea; FS = frank sickness

Note: Subscript on subjects' initials indicates order of condition.

dome was rotated around their roll axis at speeds of 30°, 45°, and 60°/sec. The astronauts' heads were fixed to a bite board. On Earth, the astronauts stood during the test, while in orbit, they either let their body float freely or were restrained in a standing position with elastic cords. The cords were used to create an upward force on the feet and give a proprioceptive input. Preflight, inflight, and postflight studies were done. Only subjectively determined results are available now.

It was found that both circularvection and tilt sensations were enhanced during weightlessness relative to ground results. The greatest enhancement occurred with the body floating freely. One astronaut reported that during a test in orbit, he sensed that he and the Spacelab were rotating together around a stationary dome. When elastic cords were used, circularvection and tilt sensations were reduced over those sensations felt with the body free, but were still greater than the sensations experienced on the ground. However, this proprioceptive effect from the cords was lost by the 5th day in orbit. Postflight results showed enhanced circularvection and tilt effects up to 5 days after the flight.

Preliminary conclusions are that visual orientation cues take on an increasingly important role in the adaptation to microgravity. Otolith organ importance is decreased. Localizable tactile cues may partially substitute for otolith cues early in weightlessness. After adaptation occurs, however, tactile cues no longer substitute for otolith inputs. This adaptation is consistent with the Sensory Conflict Theory (6).

The second Spacelab study looked at semicircular canal function in orbit. To investigate canal function, standard caloric tests were run preflight, in flight, and postflight. A caloric test involves irrigation of the external ear canal with water or air which is colder or warmer than body temperature. The test induces nystagmus with a fast phase toward the opposite ear if cold water or air is used, or toward the same ear if warm stimulation is used. The classic explanation for this phenomenon is that the temperature changes induced in the temporal bone cause circulation of the endolymph within the canals by a thermal convection mechanism. This endolymph circulation deflects the cupulae, which in turn stimulate the ampullar hair cells, leading ultimately to nystagmus.

The Spacelab 1 caloric results were quite unexpected. Early in the mission, insufficient nystagmus was generated with the caloric test to give quantifiable results. However, by late in the mission, quantifiable nystagmus was generated. A sample of these results is shown in Figure 10. We can easily see that there was no real difference in the velocity or direction of nystagmus either preflight, inflight, or postflight. These results are not what one would expect on the basis of the convection current explanation of caloric tests. Convection currents are caused by the movements of different density fluids due to gravity (14).

CONCLUSIONS

Space motion sickness has symptoms not unlike Earth-bound motion sickness; it develops rapidly in orbit and resolves over 4 to 6 days.

Space motion sickness involves some known and unknown neural pathways. The known pathways are summarized in Figure 11 (4).

The Sensory Conflict Theory is more consistent with experimental results than the Overstimulation/Fluid Shift Theory.

Many unknowns remain.

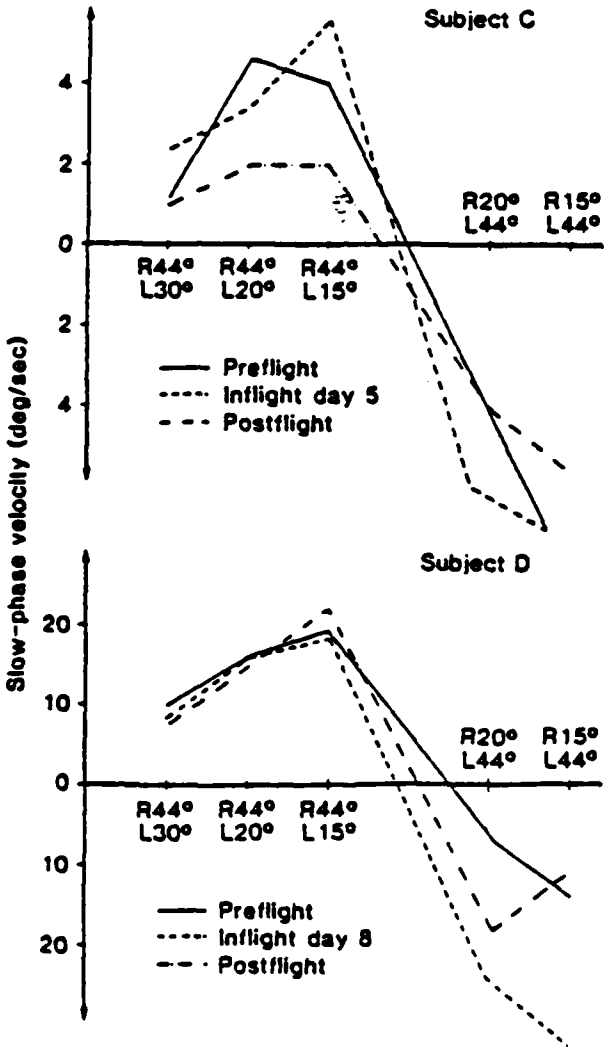

Figure 10. Nystagmus data.

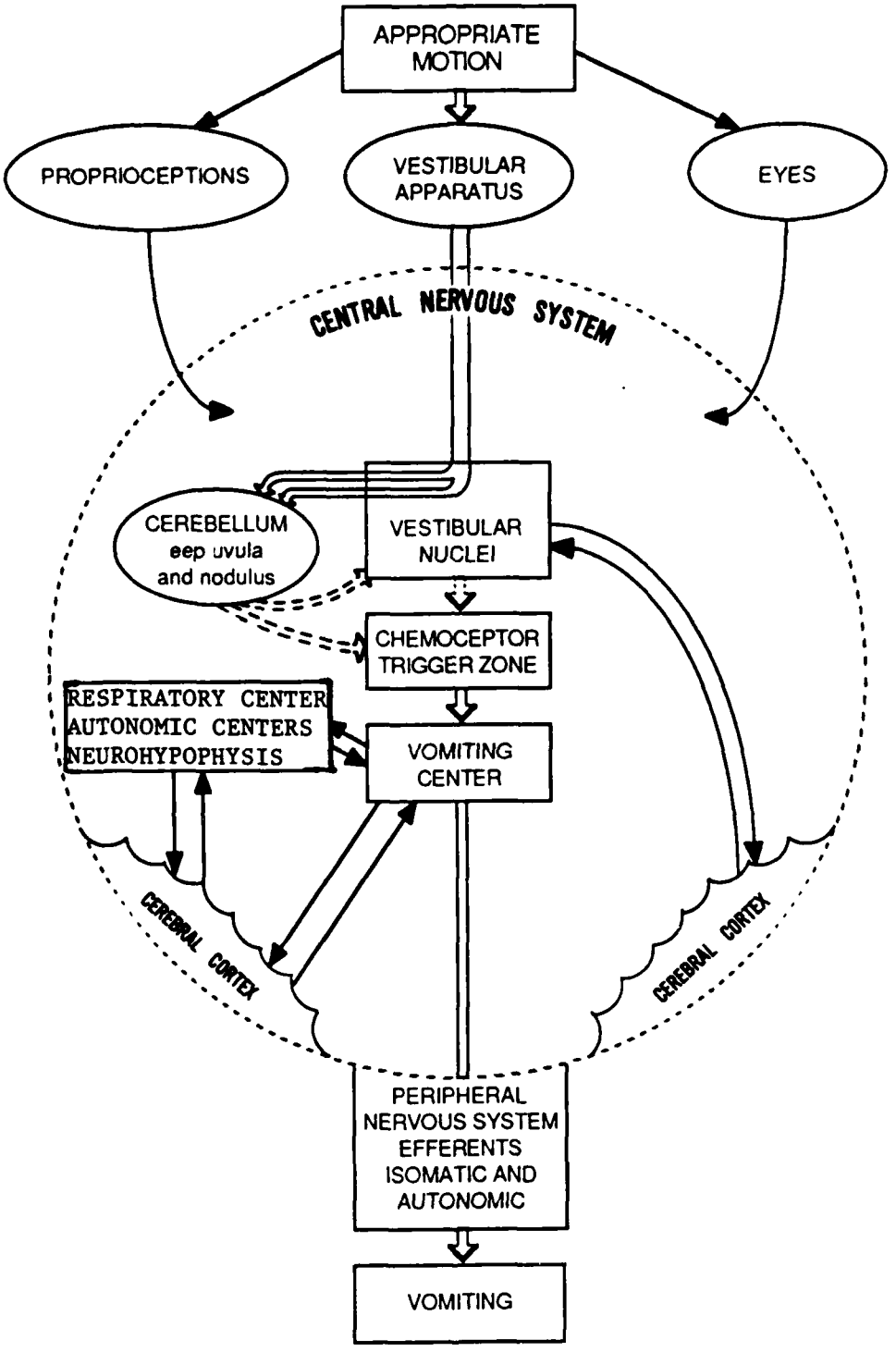

Figure 11. Neural mechanisms in motion sickness.

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